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A CONTRIBUTION TO THE PATHOLOGY OF
HEMIANOPSIA OF CENTRAL ORIGIN (COR-
TEX-HEMIANOPSIA).

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THE importance of hemianopsia with reference to the recently developed doctrine of the localization of functions in the cerebral cortex is so great as to merit the closest study on the part of the physiologist and the practical neurologist. Few subjects of such apparently small intrinsic importance have attained to such a magnitude as this one, rendering quite impossible to treat of it fully in a paper for an ordinary Society meeting. Only one or two of its phases can be considered within the time allotted me, and I shall therefore limit my remarks to the relation of the symptom hemianopsia to certain central or cerebral lesions, and endeavor to show its value for purposes of diagnosis in actual practice, rather than develop its significance for the solution of physio-psychical problems.

My apology for presenting the subject is that during the past winter it was my fortune to observe a typical case of lateral hemianopsia, stationary till the patient's death many months afterward, and almost unaccompanied by other cerebral symptoms. The topographical diagnosis made during life was verified by the post-mortem examination, so that,

apart from its scientific interest, I may venture to submit the case as an encouragement to the making of positive diagnoses *intra vitam*, in the light of the rapidly growing laws of so-called cerebral localization.

Before relating the case and presenting the specimen I should make a few remarks upon the scope of the paper, and briefly state certain data relative to parts of the subject which I cannot treat in full.

First, then, as to the scope and plan of the paper. I shall consider only the recorded cases of hemianopsia in which the autopsy revealed a lesion in some part of the brain inclusive of the optic thalami. Since the publication of Dr. Starr's valuable *résumé* of cases of hemianopsia in January, 1884, their number has somewhat increased, and I am able to tabulate forty. I should add that I have endeavored to obtain the original essay in each case, and have carefully prepared the abstracts myself: only one, No. 0, by Prévost of Geneva, has been impossible to obtain, and I quote it upon Westphal's authority, but exclude it from my tables. This labor I was induced to perform in order to avoid errors which otherwise easily occur in quoting cases, and also to be able to group these cases and more fully appreciate and present their pathological and diagnostic value. It is far from me to claim that my collection is absolutely perfect, but it is, I believe, almost complete and reliable in its critical arrangement. Let me repeat that I am anxious to present this relatively very rich and singularly harmonious collection of cases in such a way that it shall prove of most use to the practising physician for diagnostic purposes.

Second, as to the subject of hemianopsia in general.

The fact that a person might temporarily or permanently see only one half of objects placed directly in front of him has been known to physicians more than one hundred years. In 1723 Vater and Heinecke described three cases under the name of *visus dimidiatus*.

The same phenomenon was designated as hemiopia at the close of the last century, probably first by A. G. Richter, a term which prevailed and is still employed though in a dif-

ferent sense since the introduction of hemianopia by F. Monoyer in 1865, and of hemianopsia by J. Hirschberg in 1877. The latter is the preferable and preferred form.

As the terms are now accepted, hemiopia signifies loss of perceptive power in one lateral (or vertical) half of the retina, while hemianopsia means obscuration of one lateral (or vertical) half of the visual field. As rays of light cross within the eye before reaching the retina, it follows that, for example, right hemiopia is equivalent to left hemianopsia; or, in other words, that nasal hemiopia corresponds to and causes temporal hemianopsia.

In describing cases at the present time, it is customary and preferable to omit all mention of the retinal condition, or hemiopia, and to describe the hemianopsia, or the state of the visual field as determined by the perimeter or by ruder though sufficient tests.

Several varieties of hemianopsia are recognized.

1. *Horizontal*, superior, or inferior hemianopsia, almost always due to defects within the eye, and of relatively small interest to the neurologist.

2. *Vertical* hemianopsia, almost always due to lesions of the retro-ocular nervous visual tract, and hence of great importance in neuro-pathology. Quite a number of terms have been employed to designate the varieties of vertical hemianopsia. Of these we recognize and adopt :

- (a) Temporal hemianopsia.
- (b) Nasal hemianopsia.
- (c) Lateral hemianopsia, often designated as homonymous hemianopsia.

The first two varieties are exclusively caused, as far as our present knowledge goes, by lesion of the optic chiasm, of its lateral, or of its frontal or caudal borders.

The last variety, lateral hemianopsia, is always produced, as far as our present knowledge goes, by lesions of one optic tract, or of the more central parts of the optic apparatus as far caudad as the cortical centre for vision in one hemisphere.

The object of this paper is to study the recorded cases of lateral hemianopsia, with autopsies, due to lesions situated

in the more caudal parts of the optic apparatus, its central portions, from the primary optic centres (*lobi optici, corpora geniculata lateralia*) to the cortical visual centres, of areas.

With reference to all three forms of hemianopsia, I shall assume the following propositions as established:

1. The fact of a semi-decussation in the optic chiasm or man has been proven chiefly by the researches of von Gudden. According to these recent views (which are in part a return to the ancient theoretical statements of Newton, Wollaston, Müller, Hannover, and von Graefe) the optic fasciculi are disposed as follows: The fibres of each tractus opticus at the chiasm divide into two parts: A larger one which decussates with its homologue and enters into the composition of the opposite optic nerve, supplying the nasal half of the retina. This is the fasciculus cruciatus. The other, smaller set of optic-tract fibres does not decussate but passes on directly to form a part of the optic nerve of the same side, supplying the temporal half of the retina. This is the fasciculus lateralis. Thus each retina receives nerve fibres from both optic tracts, or, in other words, each optic tract contains fibres destined for both retinæ.

The inter-retinal fasciculus of Hannover is purely imaginary; there are no such fibres. The posterior loop of Hannover is now known, since the experiments of von Gudden, to be composed of non-optic fibres; it is the inferior cerebral commissure.

2. The connection of the optic tracts with the corpora geniculata lateralia and the *lobi optici* (anterior corpora quadrigemina) is an intimate one, but probably (in man) more for trophic and reflex purposes than for vision. Whether mere perception of light (as an excitant) may take place in these bodies after removal of the hemispheres, is still an open question. Certainly sight, in the ordinary meaning of the term, is impossible under such conditions.

3. A total lesion of one tractus opticus fatally produces lateral hemianopsia of the fields opposite the lesion.

4. A lesion acting upon one side of a tractus opticus so as

to compress only some of its fibres will produce one-sided nasal hemianopsia.

5. A lesion acting simultaneously on the sides of the optic chiasm will, by injuring both fasciculi laterales, produce nasal hemianopsia in both eyes.

6. A lesion compressing the optic chiasm in its frontal or caudal borders will produce bilateral temporal hemianopsia by injury to both fasciculi cruciati.

7. All such lesions are apt to be accompanied by pupillary irregularity or immobility, by optic neuritis or atrophy; and their diagnosis is further facilitated by finding signs of paralysis of other basal nerves, or of crossed hemiplegia.

8. It must be borne in mind that a lesion of the hemisphere may be so situated as to press downward upon one tractus opticus, and thus produce hemianopsia of the peripheral type (see case of Hirschberg, No. 5).

9. Lesions of the lobi optici in man have been rarely observed, and when observed have been bilateral in their effects, so that nothing can be said at present of hemianopsia due to disease of these parts.

With this brief introduction I now pass on to the consideration of the clinical and pathological aspects of the subject of my paper as exemplified in forty cases with autopsies, and five traumatic cases without autopsies, which I have been able to collect.

After a careful analysis I have grouped these forty-five cases into six categories.

1. Cases which are indefinite or useless for the study of localization, four in number.

2. Cases of lesions of parts which we have good reason to believe unconnected with the central optic apparatus, and which produced hemianopsia by pressing upon the optic tracts or the chiasm, three in number.

3. Cases in which the hemianopsia was due to a lesion of the corpus geniculatum laterale or the thalamus opticus, or both, six in number.

4. Cases of hemianopsia due to lesion of the white substance of the occipital lobe, eleven in number.

5. Cases of traumatic hemianopsia, due to injuries of the

occipital region of the skull and lesion of the subjacent brain, five in number.

6. Cases of hemianopsia due to lesions of the cortex of the brain, cortex only, or also of the subjacent white substance, sixteen in number. In this class I have included my own case. Among these sixteen cases there are four (Nos. 28, 29, 41, 45) in which the lesion was circumscribed, and where it occupied so nearly the same spot in the cortex cerebri as to afford us, in my opinion, a solution of the problem of the location of the cortical visual centre in man.

In order to shorten this essay for publication I have tabulated the cases according to the above grouping of the cases.

The four conclusive cases I shall, however, offer in full abstract illustrated with diagrams in order to enable the reader to more fully appreciate their value. But first I shall give the details of a traumatic case which is of extreme interest, from the facts that hemianopsia has existed as the sole symptom for twenty-three years, and that the cicatrix in the head is so distinct as to allow of study at present.

CASE 3.—Keen and Thomson. P. H., a soldier, aged twenty-three, was wounded in the head by a minié ball, during the battle of Antietam, in September, 1862. The missile entered the skull in the median line, $1\frac{1}{4}$ inches above the external occipital protuberance, and made its exit at a point 2 inches distant from the median line, and 3 inches distant from the point of entrance. There was no immediate loss of consciousness. In the next few days the patient complained of impaired vision. Ten days after the injury, loss of consciousness, with right-sided hemiplegia, occurred. Paralysis and imperfect memory lasted for two or three months. Apparently no aphasia.

When seen by the authors, in 1870, there was no paralysis, and the mental functions were unimpaired. The patient complained that the vision of his right eye was deficient. The pupils, ocular muscles, and fundus were normal. The left cornea bore an old opacity. Central vision on right side, = 1; on left side, $\frac{2}{3}$. The fault complained of by the patient was found to be a complete right lateral hemianopsia, with a vertical division line.

Recently I traced this soldier, through the Pension Bureau at Washington and the office at Philadelphia, to his home in that city. He has consented to come here this evening, in order to make the report more exact. By the courtesy of Drs. Keen

HEMIANOPSSA.

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TABLE I.

Cases of Hemianopsia in which the Relation between the Lesion and the Optic Defect was not Evident; Indefinite and Insufficiently Reported Cases.—Four in Number.

No.	OBSERVER.	SEX.	AGE.	FUNDUS AND PUPILS.	HEMIANOPSIA.	RELATED LESION.	OTHER SYMPTOMS.	OTHER LESIONS.	REMARKS.
20	Charcot and Pitres, 1877.	F.	52	(?)	Left lateral H. (De Wecker and Landolt.)	(?)	Localized epileptiform attacks in l. face, neck and arm. Left hemiplegia with facid muscles.	Various old patches of softening in different lial lobes, optic tracts, and chiasm not mentioned.	State of occipital patches of softening in different lial lobes, optic tracts, and chiasm not mentioned.
21	Linnell, 1881.	M.	63	At first exam. fundus normal; at second, right side of left disk pale. V. = $\frac{3}{10}$ or $\frac{2}{9}$.	Left lateral H.	Sclerosis (?) of right optic tract. Softening of corp. quad. et genic. (?)	Attacks of neuralgia in head and limbs for 8-9 years. "For 4 years" its centre, and wholly paralysis "atkins" in white substance, with numbness and tingling. Sudomotor. Quad. den blindness after severe headache, limb, as also the corp. provenient in V. Blindness, and ventral part of thalamus, more on left side. Right optic tract firmer than left, cliaxias and nerves normal.		
25	Petrina, 1881.	M.	53	Normal fundus and papillae. Amblyopia. V. notia of left eye: meningitis, hemorrhage, and soften- verigo. Falling sight.	No hemianopsia. Amblyopia. V. notia of left eye: meningitis, hemorrhage, and soften- verigo. Falling sight.	Pissure in right lamboid suture. Pachy-clinuit with loss of C. measured; no after using this instrument of re-eye alone a few minutes every cortex of occipital gyr. symptoms. All special thing becomes especially the 2d and 3d, senses normal except confused and from suicus temporisight. Died of pneumonia.	Fall backward on occiput; unconscious, and bleeding from nose, mouth, orbital arteries. Violent head- temporal lobe, tentorial foci in r. ear. Impaired mental form nucleus, medial occip. lobe and action; no paralysis. In lary sub. of frontal optic tracts, a few weeks apoplectic lobe and gyrus olfact attack with left hemi- Also in left parietal pachymeningitis; recovery. Later, lobe and left thalamus.	Atheromatous cere- bral arteries. Old hemis. not well given. No specific mention of occip. lobe and frontal optic tracts.	
40	Wiethe, 1884.	M.	54	Fundus normal. Pupils and V. not mentioned. Binocular V. preserved.	Superior lateral hemianopsia. (?)	Lesion of left thalamus opicus. (?)	Fall upon occiput; unconscious, and bleeding from nose, mouth, orbital foci in r. ear. Impaired mental form nucleus, medial occip. lobe and action; no paralysis. In lary sub. of frontal optic tracts, a few weeks apoplectic lobe and gyrus olfact attack with left hemi- Also in left parietal pachymeningitis; recovery. Later, lobe and left thalamus.	Complete (?) blindness developed in 1/4 hours; partial recovery. Death from hernia.	

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TABLE II.
Cases of Hemianopsia from Lesions Unrelated to the Cortical Centre for Sight; Cases from Pressure Transmitted to Optic Tracts, etc.—
Three Cases.

No.	OBSERVER.	SEX.	AGE.	FUNDUS, PUPILS, ETC.	HEMIANOPSIA.	RELATED LESION.	OTHER SYMPTOMS.	OTHER LESIONS.	REMARKS.
5	Hirschberg, 1875.	M.	40	fundus normal, Central H. with sharply defined vertical line passing close to point of fixation.	Left tractus opticus smaller than right.	For four years severe left-sided, intermittent headache. Imperfect V. to right. Right hemi- plegia. Aphasia.	Glio-sarcoma, size of an apple, in left frontal lobe.	Tumor may have pressed on optic tracts. State of occipital lobe not mentioned.	
8	Huguenin, 1876.	F.	46	(?)	Right lateral H. with a not well defined vertical line.	Attack of uncon- sciousness; right hemi- plegia with partial an- esthesia; aphasia, alex- ia, and word-deafness. (?)	Embolism of 1. mid- cerebral artery; so-called tening of Broca's gyrus; gyrus precentralis, et postcentralis, in their lower parts; insula, ex- ternal capsule, clau- strum, and external di- vision of N. lentiformis.	State of occipital lobe not mentioned.	
16	Pflüger, 1878.	M.	62	(?)	Left lateral H.	Injury of inferior part of thalamus by clot, hemorrhage. with pressure on tractus. (?)	Attack of cerebral hemorrhage in corpus striatum, and inferior part of thala- mus.	In right lateral ven- tricular much semi-fluid blood. Hemorrhage in mentioned.	

TABLE III.

Cases of Hemianopsia from Lesions of the Thalamus Opticus and Corpora Geniculata.—Six Cases.

No.	OBSERVER.	SEX.	AGE.	FUNDUS, PUPILS, ETC.	HEMIANOPSIA.	RELATED LESION.	OTHER SYMPTOMS.	OTHER LESIONS.	REMARKS.
6	H. Jackson and W. R. Gowers, 1875. Pooley, 1877.	M.	65	(?)	Left lateral H.	Softening of caudal part of right thalamus. Optic nerve disintegrated.	Left hemiplegia and hemianesthesia. Death from a non-cerebral disease.	In left occipital lobe there was a "gummy tumor," 12×30 mm., ad- mors.	Syphilitic ar- thritis.
9		M.	55	V. normal when first seen. Later, choked disk in left eye, lateral H., passing away, but returning in a few days.	Fields normal right, surrounding white substance.	Complete softening of left thalamus and optic nerve.	For 6 years epileptic seizures, right hemiparesis, lateral ventricle much aphasic symptoms, dilated.	In left occipital lobe there was a "gummy tumor," 12×30 mm., ad- mors.	
19	Dreschfeld, Case I., 1880	F.	41	(?)	Left lateral H.	A tuberculous tumor almost replaced right thalamus, and extended laterad through int. capsule to nucleus opticus, lenitiformis, tractus opticus reduced to a thin band.	Paroxysms of headache and weakness of left leg for 7 years, almost to the surface of brain.	The tumor extended to a thin band.	
30	Dreschfeld, Case II., 1882.	M.	40	Fundus normal. Central V. good.	Left lateral H.	Cysto-sarcoma in place of lateral part of right thalamus; optic nerve and tractus opticus, internal capsule, part of nucleus opticus involved.	Headache, giddiness, transient aphasia. Left hemianesthesia, hemiplegia.	Several clots within body, Special senses normal except V.	
31	Dreschfeld, Case III., 1882.	F.	52	Pupils equal and react well. Central V. fairly good.	Left lateral H., with a vertical line not quite reaching point of fixation.	Optic nerve tumors in post. seg. of both lobes.	Bright's disease and extremitiy of right thalamus (puvinate), optic tracts, optic nerve, and optic nerve.	Right hemiplegia, right amblyopia, episodic convulsions, transient aphasia. Also one under the corpus callosum.	
36	Rosenbach, 1883.	F.	34	(?)	Right lateral H.	Softening of external part of left thalamus; tumors in post. seg. of both lobes.	Softening of corpus callosum and internal capsule.	Atrophy of left part of mental tractus opticus and of right optic nerve. Symptoms of pressure.	

TABLE IV.
Cases of Hemianopsia from Lesions Situated Chiefly in the White Substance of the Occipital Lobe.—Eleven Cases,

No.	OBSERVER.	SEX.	AGE.	FUNDUS, PUPILS, ETC.	HEMIANOPSIA.	RELATED LESION.	OTHER SYMPTOMS.	OTHER LESIONS.	REMARKS.	
2	Lewick, 1866.	M.	40	(?)	H., of which lobe not stated, when first seen, sphere, $\frac{1}{4}$ inches diameter (Pepper).	Abscess in posterior lobe of right hemi- sphere, by falling ob- jects. In July and Au- gust, headaches and ex- abscess in anterior lobe of left hemisphere. Lat- tice, sense of feeling erat drunk. No convulsions lined by drum. Death in low inflam- matory. Death in low inflam- matory.	In May and June; Opacities in arachnoid. Slight (?) injuries various structures at base of brain softened and discolored. Small gout, headaches and ex- abscess in anterior lobe of left hemisphere. Lat- tice, sense of feeling erat drunk. No convulsions lined by drum. Death in low inflam- matory. Death in low inflam- matory.			
4	Hosch, 1879.	M.	54	V. R. L. $\frac{1}{4}$ g. redness	Left lateral H., with verti- cal redness and cal line a little below of pa- pillae, and a few streaks of hem- orrhage in ret- ina.	Large hemorrhagic cyst, almost destroying lateral occipital lobe of point of cortex.	Large pigmented cic- atrix in right corpus callosum, extending into darkening of fields of thalamus. N. caud. et V. Complete left hemi- labyrinth, atrophied. Last attack due to a large hemorrhage in 3d ventricle. In fourth fresh attack, right side par- alyzed; death.	In centre of right thalamus was a so- called apoplectic cica- trix, half size of a lentil. Tracts optici and op- tic nerves normal. Fatty heart and con- tracted kidneys.		
13	Baumgar- ten, 1878.	M.	(?)	Central normal.	V. H. suddenly developed and persistent.	In the substance of the right occipital lobe was an old hemorrhagic cyst as large as a wal- nut, and the various control of the occipital lobe were softened, though recognizable.				
15	Dmitrov- sky and Lebeden, 1879.	R.	22	Papilla con- gested obscure H.	Right lateral	In the left cerebral hemisphere, a clot which involved the greater part of the cor- ona radiata, and pen- etrating the temporal lobe almost to the cor- tex.	Headache, drowsi- ness, a clot which involved the speaking. Aphasia.			

TABLE IV.—*Continued.*

No.	OBSERVER.	SEX.	AGE.	FUNDUS, PUPILS, ETC.	HEMIANOSIA.	RELATED LESSONS.	OTHER SYMPTOMS.	OTHER LESIONS.	REMARKS.
22	Westphal, Case I., 1881.	M.	42	Slight neuritis in eye.	Left optic nerve in I.	Old focus of softening in white subst. of brain, passing between parietal and occip. lobes, as 1. line through point of fixation. temp. gyrus.	Left-sided convulsions, followed by hemiplegia.	Basal optic apparatus and thalamus normal.	
24	Senator, 1881.	M.	69		(?) H.	Clot occupying greater part of white subst. of right temporal lobe, deviation to right; outside inf. horn of speech normal.	Thalamus normal; constrictor right corp. genic. lat. to right; flattened and yellowish- red. The clot also ex- tended frontoid through ventricle.		
32	Stenger, Case VII., 1882.	M.	32	At close of life, for two months, blind-repeated tests.	Left lateral horn, is a distinct, rather broad furrow, extend- ing from post. border of thalamus	In white substance bordering right post. tia, paralytic; tremors, attack of hemispasm or hemiplegia. Later, attacks of left-sided spasm.	Symptoms of demen- tia, paroxysms, thick whole of both occip. lobes, which are short- ened and shrunken.	Decortication over left parietal lobe and post. border of both hemispheres.	

TABLE IV.—Continued.

No.	Observer.	Sex.	Age.	Fundus, Pupils, etc.	Hemianopsia.	Related Lesion.	Other Symptoms.	Other Lesions.	Remarks.
34	Wernicke and Hahn, 1882.	M.	45	No lesion in fundus.	Right lateral H. Vertical line subst. of passing a little sphere, latero-dorsal of point of fixation.	Abscess in white hemi. In I. frontal and occipital regions; a cloud meningitis.	Chronic phthisis, pain in front. before right eye. Mind was opened. The caudal lobe was almost lost.	Ventricle perforated and containing pus. No abscess of occip. lobe was made.	The diagnosis of meningitis and con- tritum and surgically the case is most encouraging.
35	Jany, 1883.	F.	21	V. much re-duced. R. + H. S. = 5°. L. 10°. Fingers counted at 8°.	Right lateral H. Vertical line a little outside tumor lies at apex of fixa-tion. Neuro-retinitis. passing into outer field con- atrophy, more tracte-d in r. eye.	Cysto-sarcoma occur-ping nearly whole of occip. lobe.	Severe headache. Cystic part of tumor.	Severe headache. Cystic part of tumor.	
36	Richter, 1883, Case I.	M.	54	Pupils equal, normal in size H. Vertical di- and action. Optic nerve pale; vessels smaller.	Left lateral H. Vertical di- and vision line.	Clot of a certain age in r. occip. lobe in noia. White subst. of V. separated from it by ependyma. Cortex uninjured.	Halucinatory para-esthesia in left eye, weak, temporal lobe, just la-and left hand numb. Third and fresh clot separated from it by ependyma. Cortex	Most ancient clot in temporal lobe.	
37	Schmalz, Cited by Vetter, 1883.	F.	69	Pupils and ocular muscles H. normal.	Right lateral H.	Yellow softening in left hemisphere, most destructive in occipital lobe. Caudal part of thalamus softened.	Apoplectic attack, hemiplegia, complete r. hemi-brain; pre- and post-central, parietal, and sense lost. Left arm occip. gyri.	Yellowish softening of various gyri in l. hemisphere, most followed by r. hemiplegia. Complete r. hemi-brain; pre- and post-central, parietal, and sense lost. Left arm occip. gyri.	

TABLE V.
Cases of Hemianopsia due to Injuries to the Cranium and Brain; Lesion mostly Cortical.—Five Cases.

No.	OBSERVER.	SEX.	AGE.	FUNDUS, PILLS, ETC.	HEMIANOSIA.	RELATED LESION.	OTHER SYMPTOMS.	REMARKS.
3	W. W. Keen and W. H. Thomson, 1871.	M.	32	V. R. = 1. L. $\frac{3}{2}$. Opacities H. Anterior cornea. Pupils, ocular motions, fundus normal.	Right lateral wound of the cranium on left side. Ten days later loss of consciousness followed by r. hemiplegia. No above the external occipital protuberance. Imperfect memory last- ing 2-3 months. Exit 2 inches (50 mm.) from median line, and 3 paralysed in 1870. (75 mm.) from en- trance.	In Sept., 1862, re- ceived a gunshot wound of the post. end of paired V. soon noticed entrance in median consciousness followed line $\frac{1}{4}$ inches (31 mm.) by r. hemiplegia. No above the external occipital protuberance. Imperfect memory last- ing 2-3 months. Exit 2 inches (50 mm.) from median line, and 3 paralysed in 1870. (75 mm.) from en- trance.	No immediate loss of consciousness. Im- pulsive acts. Im- pulsive acts. Im- pulsive acts.	Hemianopsia caused by in- jury to optic fasciculus. Oc- cipital gyri un- injured.
<i>Idem.</i>	Observed by E. Seguin, in 1885.	M.	46	Pupils normal. Eye mus- cles normal, not quite reach- is blood- vessels normal. Fundus: blood- vessel normal. outer temporal quadrant of both discs whiter than right. V. R. = $\frac{2}{16}$ with — $\frac{7}{12}$; ax. 90°. V. L. = $\frac{4}{16}$ with — $\frac{7}{12}$; ax. 90°. R. reads No. 14 J. at 12° with + $\frac{1}{15}$ S. No. 1 at 8°. L. reads No. 14 J. at 12° with + 1 S.; No. 6 at 8°.	Entrance cicatrix barely noticeable. Exit tack 6 years ago (1870). A large cup-shaped Right side a little weaker; more easily affected by alcohol. Bridge of bone. Uses left hand habitu- ally. Mental action between entrance and exit only 3 cent. Both good.	One epileptiform at- tack 6 years ago (1870). Injury initiated on the cadaver shows an in- jury of parietal lobe, dorsal of angular gyrus. Occipital gyri uninjured. White sub- stance deeply lacer- ated.		

TABLE V.—*Continued.*

No.	Observer.	Sex.	Age.	Fundus, Pupils, etc.	Hemianopsia.	Related Lesion.	Other Symptoms.	Other Lastings.	Remarks.
4	Hughes, 1873.	M.	38	Eyes normal except hemianopsia. (Prof. at time of discharge from Drs. Wilson, Jacob and Swansy of Dublin.)	Right lateral hemianopsia.	Injured by fall of a large iron vessel on back part of head.	Coma relieved by operation.		
20	Schmidt-Rimpfert, 1881.	M.	(?)	?	Left lateral H.	Vertical compound fracture of right occipital and parietal bones. Several pieces of bone removed and com relieved at once. Complete recovery in 3 mon., except impairment of V.	No paralysis, covered with complete deafness of r. ear; left by a fall. A handful of lat. H., and occasional bony splinters and some dizziness with loud tinnitus in r. ear.		
23	Heuse, 1881.	M.	(?)	Slight opacities of cornea, H. Right temporal, reducing V, and nasal half-fields rendering ophthalmoscopic examination unsatisfactory.	Right lateral hemianopsia.	Fall upon head causing a depressed fracture of left side of cranium. Enormous nasal half-fields cicatrical depression of bone on the left side of the occipital end of the skull; a strong ridge-like depression extended from left parietal into the occipital bone.	Paralysis of right side, falling down steps.		
37	Nieden, 1883.	F.	22	During second paralytic seizure before H. temporary loss of eye sight.	After operation.	At operation, 9 months after injury, severe headache, vomiting, pain in left eye, vomiting, pain in inner occipital region.	Paralysis with partial anesthesia of right side.		This case was probably one of hysterical nature, and even the optic symptoms may have been of same sort.
				Fields normal, (nasal) quadrigeminal, Ninth nasal areas.		At operation, 9 months after injury, severe headache, vomiting, pain in left eye, vomiting, pain in inner occipital region.	Paralysis with partial anesthesia of right side.		
				Slight venous dilatation of left eye limited but the pupilae and in its upper and lower retina.		Hyperemia of left eye limited but the pupilae and in its upper and lower retina.	Paralysis with partial anesthesia of right side.		
				Ninth nasal areas.					
				Day after operation V.L. = 49.					
				Field normal, V.R. = 26.					
				Temporal field wanting to median line.					

TABLE VI.

Cases in which Lateral Hemianopsia was due to Lesions of the Cortex of the Brain and Subcortical White Matter (mostly Cortical Lesions).—Sixteen Cases (Arranged in the Order of their Value for Studying the Localization of the Visual Centre).

No.	AUTHOR.	SEX.	AGE.	FUNDUS, PUPILS, ETC.	RELATED LESION.	OTHER SYMPTOMS.	OTHER LESIONS.	REMARKS.
1	Chaillou, 1863.	F.	69	(?)	Lateral Side not stated; most probably of Sylvius and of inner covering corresponding white matter of occipital lobe much atrophied. with hemianopsia observed	Atrophy of gyri at the end of left (?) fiss. face of occipital lobe; V. and weakness of r. arm. Previous to ad- mission patient had had 5 cent. Right hem. of mus must have been in "right hemisphere". infect contained a cavity 2 x 1 cent. leaving "left hemisphere". Hemianopsia. older still. Death in a third attack.	Small patches of soft- ening in left (?) thala- mus. Inferior part of sella in autopsy. Left corpus striatum. Lesion of occip.	Sides of brain probably rever- sal of sella in autopsy. Left corpus striatum. Lesion of occip.
26	Westphal, Case II., 1882.	M.	38	R. R. pupil II. trifle wider. lune, 1880.	Central V. nor- mal. R. pupil II. discovered angularis, eye - muscles. Nearly vertical over cuneus and pre- and imperfect speech. not stated. No line passes a cuneus. Optic nerves in August, awoke with lesion to optic little to left (?) and chiasm normal. of point of fixa- tion.	Lateral softening of gyrus angularis, of occipital lobe (?) convulsions, showed an extensive softening, involving post - central area, as far as supra-marg. r. hemiplegia (arm gyrus, the whole most) of complete parietal lobe, gyrus an- terior, word-guiars, and nearly the deafness and amnesia whole of occipital lobe. aphasia. Later, repeat- ed convulsions (clonic) on r. side. Slight r. and 3d temp. gyri. No paresis; great loss of muscular sense.	In June, 1879, also Left, he misper- ceived objects here convulsions, followed by delirium, cortical softening, involv- ing post - central area, as far as supra-marg. r. hemiplegia (arm gyrus, the whole most) of complete parietal lobe, gyrus an- terior, word-guiars, and nearly the deafness and amnesia whole of occipital lobe. aphasia. Later, repeat- ed convulsions (clonic) on r. side. Slight r. and 3d temp. gyri. No paresis; great loss of muscular sense.	Admitted with de- mentia paralytica. Cerebral soft- ening over right parietal and occipital lobes; fasciculus to body, followed by 1. subjacent white subst., thalamus.
32	Stenger Case VIII., 1882.	M.	52	Special senses normal on ad- mission.	L. lateral hemi- anopsia after left hemipar- esis. Persistent of Gratiolet.	Softening of white subst. of r. par. interrupting fasciculus of Gratiolet.	Int. and ext. hydro- cephalus. Cortical soft- ening over right parietal and occipital lobes; fasciculus to body, followed by 1. subjacent white subst., thalamus.	Degeneration from visual cor- tex along optic tract to optic nerve, and much atrophy. Right thala- mus smaller and softer than left. Vary- ing l. hemiparesis. Death in general con- vulsions.

TABLE VI.—Continued.

No.	Author.	Sex.	Age.	Fundus, Pupils, etc.	Hemianopsia.	Related Lesion.	Other Symptoms.	Other Lesions.	Remarks.
7	Förster and Wernicke, 1876.	M.	—	(?)	At first, temp. field of right eye obscured (Sylvius, vertically aligned, parietal lobe and nearest to point of gyrus angularis, pene- fixation; the nasal field of left ventricle, probably in- was darkened, terminating the optic also almost to fasciculus. Later, a degree of left lateral hemianopsia was found, but the original lim- its of the right hemianopsia re- mained un- changed.	The area of softening of right caudad of the fissure of Sylvius, including the aphasia.	Repeated paralytic attacks on right side;	Embolus in left Sylvian artery, softened patch in cortex, includ- ing inf. parietal lobe, separate ac- gyrus angularis, and counts were frontal part of occipital lobe; numerous small necrotic foci in l. nu- cleus lenticularis, nu- cleus caudatus, thala- mus, and external cap- sule. Insula, chiasma, and optic nerves normal.	This case has been sometimes published twice, as infl. parietal lobe, separate ac- gyrus angularis, and counts were frontal part of occipital lobe; numerous small necrotic foci in l. nu- cleus lenticularis, nu- cleus caudatus, thala- mus, and external cap- sule. Insula, chiasma, and optic nerves normal.
10	Jastrowitz, Case I., 1877.	M.	(?)	Transient pa- ralysis of R. N.V.I. Papilla normal; V. re- lative good; fixation pos- sible.	Right lateral hemianopsia (Hirschberg).	Soft sarcoma of left occipital lobe, involv- ing occip. gyri and precentral precuneus. It extended ataxic, aphasia, agraphia. Right hemiplegia appeared as far laterad later; paralysis of vari- which presented a slight discoloration and punctate hemorrhages as far as the thalamus.	Vertigo; loss of mem- ory, of energy; general precuneus. It extended ataxic, aphasia, agraphia. Right hemiplegia appeared as far laterad later; paralysis of vari- which presented a slight discoloration and punctate hemorrhages as far as the thalamus.	Thalami, lobi optici, tractus, chiasma, and nervi optici presented no alterations.	Drank sulphuric acid with usual local effects. There occurred on 10th day, embolism of right inner coat of aorta, brachial artery. A few days later patient com- plained that he could not see well with his left eye. No other symptom of local dis- ease in brain. Death, of inanition, on 16th day.
18	Cursch- mann, 1879.	M.	50	(?)	Left lateral H., which per- sisted from 10th to 16th day (death).	Large focus of soft- ening in right occipital surface, especially on its caudal and mesial parts.	Usual lesions of reso- phagitis, etc. An ex- tension of inflam- mation of right inner coat of aorta. Complete embolism of right brachial artery.		

TABLE VI.—*Continued.*

No.	AUTHOR.	SEX.	AGE.	FUNDUS, PUPILS, ETC.	HEMIANOSIA.	RELATED LESION.	OTHER SYMPTOMS.	OTHER LESIONS.	REMARKS.
11	Jastrowitz, Case II., 1877.	(?)	(?)	(?)	Limitation of visual fields to the right.	Large patch of soft- ening in left occipital lobe.	Aphasia.	Partial embolism of left internal carotid artery.	
17	Nothnagel, 1879.	M.	51	(?)	Partial right lateral H. (besides several shortly before lesions) death, total loss of blindness.	Patient awoke with partial paralysis of left hemisphere (there was a yellowish-red softening of the entire occipital lobe). Chiasma, optic thiasa, tracta, and nerves normal.		Carcinoma of pan- creas, with various sec- ondary deposits. En- cephalitis aortica ver- rucosa. Numerous spots of embolic soften- ing in brain. In r. hem. a patch in middle of precentral and post- central gyri, softening of caudal extremity of sup. parietal lobe, with extension into in- traparietal fissure, and into white substance to ventricle. Small patches in r. occipital lobe.	
27	Marchand, 1882.	M.	72	(?)	Left lateral H. — "complete," (Dr. Pepinflor)	Patch of softening in right occipital lobe; apex of occip. lobe occupied by a necrotic patch as large as a hazelnut, separat- ed from deeper parts of brain by a softened yellowish zone about .5 cent. thick.	Sudden left hemip- legia. Death in a few months. No details as to motor and sensory symptoms.	Senile dementia.	Gyri adjacent to occipital lobe.
42	Richter, Case II., 1885.	M.	70		After H. there occurred con- junctivitis, ker- atitis, cataract, phthisis of left eye.	Complete left lateral H. Defect persisted in right eye.	Patch of softening in right occipital lobe.	Syphilis in 1865. Since 1880, repeated apoplectic attacks, accompa- nied by temporary r. of Reil. hemiplegia and aphasia.	
43	Richter Case III., 1885.	M.	48		L. pupil re- acts well; right H. Very slightly. Left optic nerve normal; right, atrophied.	Left lateral Patch of softening in right occipital lobe.		In 1882, l. hemiplegia and hemianopsia. De- mentia.	Small psammoma in r. optic nerve. Patch of softening in l. island of Reil.

TABLE VI.—*Continued.*

No.	AUTHOR.	Sex.	AGE.	FUNDUS, PUPILS, ETC.	HEMIANOSPIA.	RELATED LESION.	OTHER SYMPTOMS.	REMARKS.
44	Richter, Case IV, 1885.	M.	40	Pupils react well. Fundus suddenly de- normal. Mus- tained in los- over cuneus, and lower part of fundus normal. Pupil limit to temporal gyrus. Tip of gait, weak legs, tongue tremulous, speech slurred. Granular bodies found thick. Hallucination of white substance of V. Death from epilep- toid seizures.	Left lateral H. Firm adhesion of pia to occipital gyrus, also leptoform attack, men- tally and thickening of meninges. In various parts, opa- cification, loss of power of fixation. Left eye only, shorter than its fellow. Granular bodies found thick. Hallucination of white substance of V. Death from epilep- toid seizures.	Syphilis in 1869. Epilepsy. Basal nerves normal.	Basal nerves normal. In various parts, opa- cification, loss of power of fixation. Left eye only, shorter than its fellow. Granular bodies found thick. Hallucination of white substance of V. Death from epilep- toid seizures.	
45	Seguin, 1885.	M.	46	Pupils and fundus normal. Central vision persisting good with glass- opie (Dr. C. R. Agnew).	Sudden attack of left lateral H., lasting persisting till the whole of the right heart, various peripheral embolisms, pseudo- erythrocytosis, intermittent fever. No other signs or symptoms.	Mitral regurgitation, almost enlargement of the mitral valve, tissue of hemiplegia containing balls of mid-distinct ana- stomosing vessels. Large in the brain, and con- sists in kidneys and vessels makes it almost certain that there were no gross lesions on surface of brain, and with in the brain. Location of lesion cor- rectly diagno- sed during life.	Verrucose disease of the mitral valve, tissue of hemiplegia containing balls of mid-distinct ana- stomosing vessels. Large in the brain, and con- sists in kidneys and vessels makes it almost certain that there were no gross lesions on surface of brain, and with in the brain. Location of lesion cor- rectly diagno- sed during life.	
46	Haab, 1882.	M.	68	= r. Optic nerves present, but "a sensible grayish color." Central vision only, shorter than its fellow. Vertical line patch reaching quite (?) up to point mostly on mesial aspect of fixation. H. of occip. lobe, includ- ing apex. It involves the cuneus in its inferior half, the fifth temporal gyrus, and fissura hippocampi. White sub- stance destroyed as far as ventricle.	Left lateral H. Complaint of hemispace only, shorter than its fellow. Vertical line patch reaching quite (?) up to point mostly on mesial aspect of fixation. H. of occip. lobe, includ- ing apex. It involves the cuneus in its inferior half, the fifth temporal gyrus, and fissura hippocampi. White sub- stance destroyed as far as ventricle.	Endo- and peri-cardi- itis, sudden attack of lesion. Optic nerves, during life rapidly passing away almost entirely, leaving almost normal (microscopic ex- amination). No hemianes- thesia. Death in two years.	No other cerebral nerves, during life chiasma, and tracts nor- mal. No gross lesions on surface of brain, and with in the brain. Location of lesion cor- rectly diagno- sed during life.	

TABLE VI.—*Continued.*

No.	AUTHOR.	SEX.	AGE.	FUNDOS., PUPILS, ETC.	HEMIANOPSIA.	RELATED LESION.	OTHER SYMPTOMS.	OTHER LESIONS.	REMARKS.
41	Fréré, 1885.	F.	52	Ophthalmoscope showed II. Vertical line passing through the left cuneus, and right cuneus, and right hemiplegia. On fifth temporal gyrus, tribre, no motor symptoms, but slight r. hemianesthesia to pain and cold. Hearing, taste, and smell normal.	Right lateral	Yellow patch destroying the greater part and transient attack of lesions.	Nov. 2, 1883, slight	No other cerebral	Lobi optici, tractus, chiasm, and nerves normal.
29	Huguenin, 1882.	F.	8	At first visit, April 16th, vision and hearing good. Optic nerves normal. On 27th, slight optic neuritis.	Left lateral H.	At x 3 x 2.5 cent. lay on the mesial aspect of the right occipital lobe; later, headache in par-dyna of ventricles partly embedded in the oxysans, vomiting; regurgitation; firmly adherent curving convulsions; chiasm, and in both to pia. It was over the increasing dementia, fissura hippocampi, ex-bronchitis, thickened.	3. Pertussis followed by	A small tumor on the	

and Thomson, I have the additional information that a few days ago the hemianopsia was found unchanged, twenty-three years after the reception of the injury. I made an examination of the patient, Hughes, this morning, with the following results :

He presents no distinct paralysis or anaesthesia, or aphasic symptoms. His tongue deviates a trifle to the right, and the grasp of the right hand is a little less than that of the left. Dynamometer test : L, 38, 34 ; R, 35, 34. The knee-jerk is abnormally great on both sides, but equally so.

Tests reveal no anaesthesia, but the patient thinks that his tactile sensibility is very slightly dull on the right side of head and in right hand. The muscular sense, tested by knowledge of passive movements of fingers, and by different weights laid in both hands, while the patient's eyes are closed, is normal.

He habitually uses the left hand more than the right, but this is on account of his loss of vision on the right.

He adds that when under the influence of liquor his right leg and arm feel the effects first and most.

Only one epileptiform seizure is known to have occurred, viz.: an attack in the night, about six years ago. He claims that his memory is now good. It was formerly weak, but he never, apparently, had amnesia of words.

The scalp presents two cicatrices ; that of entrance very small, that of exit of the ball enormous and greatly depressed. The following are some topographic measurements with the head placed so as to have the skull resting on the alveolo-condyloid plane of Broca.

The entrance wound is in the median line, about 3.5 cent. above the external occipital protuberance. From the bregma along the median line to this cicatrix is 15.5 cent.

The exit scar is a large cup-shaped depression situated dorso-lateral of the other, near the parietal eminence. Its frontal edge is 6.5 cent. from the bregma ; its mesal edge nearly at the median line (.5 cent. distant) ; and its fronto-lateral edge is 12.75 cent. from the left tragus. Its transverse diameter is 5 cent., its longitudinal diameter 6.5 cent. Its depth is 1.5 cent. The bridge of bone between the two scars is only 3 cent. broad.

The bottom of the exit scar is very firm though not bony, and the patient is not affected by reasonable manipulation.

A rough test with a small white object at 18 inches shows right lateral hemianopsia, with line passing outside of point of fixation ; there is besides a darkened area in the left upper temporal quadrant.

Dr. G. W. Hale, House Surgeon of the Manhattan Eye and Ear Hospital, has very kindly made an examination of H.'s eyes, and made diagrams of his visual fields. He finds the following :

$R = \frac{2}{6} : \frac{2}{4}$ w. — $\frac{1}{2}$; ax. 90° :

$L = \frac{1}{2} : \frac{1}{4}$ w. — $\frac{1}{2}$; ax. 90° :

R reads No. 14 J at 12" w. + $\frac{1}{2}$ s No. 1 J at 8":

L reads No. 14 J at 12" w. + $\frac{1}{2}$ No. 6 J at 8":

Pupillary reaction, normal.

Eye muscles: no insufficiency either at 20' or 1'.

Fundus: blood-vessels of normal size; outer temporal quadrant of either disk whiter than normal, left a little whiter than right. No other lesions.

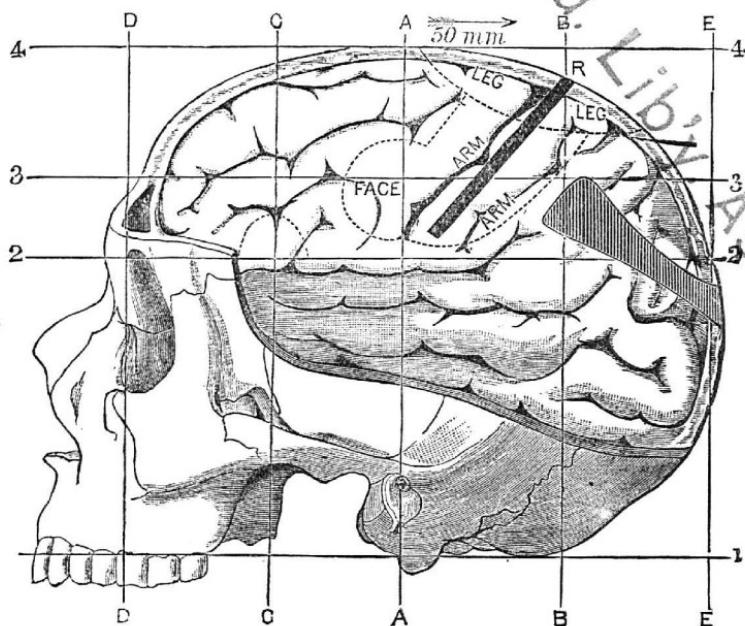


FIG. 1.—The probable course of the minié ball through the brain in case 3 (Keen and Thomson) is indicated by the club-shaped shaded figure in the occipital part of the head, extending across lines 2 and B.

By the kindness of Drs. Peabody and Ferguson I have had the opportunity of repeating the injury upon the cadaver at the New York Hospital. Trephine openings were made in the cranium of a male subject at points corresponding with the author's measurement of H.'s cicatrices, and an iron rod pushed through, followed by a large seton of jute. The hemisphere was placed in alcohol for harden-

ing. It was then found that the track of the ball was entirely dorsad of the occipital, through the parietal lobe almost to the confines of the postcentral gyrus. Its penetration was such, however, that it must have injured the optic fasciculus on its way to the cuneus. See fig. 1.

CASE 28.—Haab: male, at sixty-eight years. In Feb., 1878, while under treatment for endo- and peri-carditis, experienced an attack of paresis of the left extremities. This rapidly passed away, leaving a certain degree of disability, for after working with the left hand patient experienced pain in the left arm, and palpitation.

When seen by Haab in July the patient complained that he

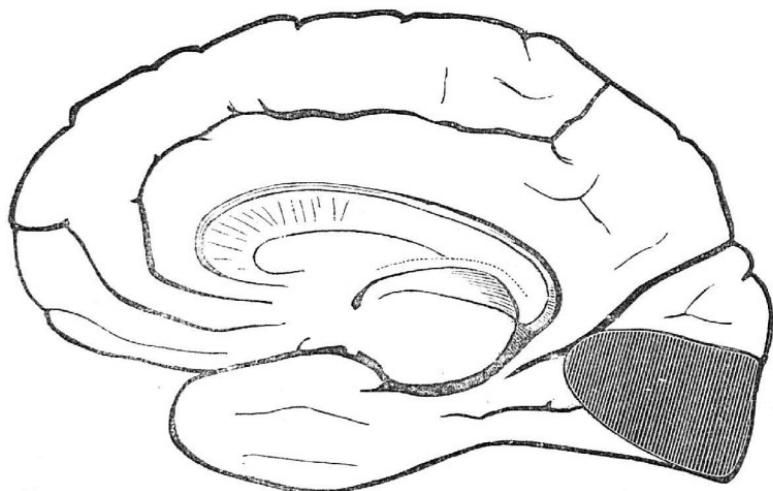


FIG. 2.—Mesal aspect of right hemisphere (Ecker) showing patch of softening in case 28 (Haab).

could not see to his left with his left eye—thought his right eye was normal. Examination showed only a trace of paresis or awkwardness in left extremities; no anæsthesia. Intelligence normal. Hearing good. Central vision = 1 (H. 2). There was left homonymous hemianopsia, the limit reaching quite (?) up to the fixation point. In right fields color-perception good.

Optic nerves present a “senile grayish color.”

During the year several re-examinations gave the same results. The patient insisted that there was a veil or cloud over the *left* eye. Death in July, 1879.

Autopsy.—The caudal end of the right hemisphere was .5 mm. shorter than its fellow. There was a depression in right occipital lobe, the pia hanging loosely over a cavity containing clear fluid. The patch was mostly upon the mesal aspect of the hemisphere (including apex). It occupied the site of the fissura hippocampi, and extended beyond it above and below. The frontal end of the cyst was at six cent. from the apex of the lobe. The white substance was but slightly injured, and there was no communication between the cyst and the posterior horn of the ventricle. The vertical height of the patch was 2-3 cent.

No other cerebral lesion. The optic nerves, chiasm, and tractus were normal to a microscopic examination. Haab's diagnosis during life was embolism of an artery supplying the hinder part of right thalamus opticus.

CASE 29.—Huguenin. A girl, æt. eight years. In autumn of 1878 whooping-cough, followed by ill-health and sluggish mental action. In January, 1879, headache in paroxysms; later, frequent vomiting, sleep broken; no motor or ocular symptoms. At end of March severe convulsions, which have frequently recurred, constituting the principal phenomenon. Increasing dementia. Seen by Huguenin, 16th April, 1879. Child demented; understands what is said, and, according to parents, replies well; memory feeble; general muscular weakness, but no localized paralysis. Vision and hearing good. Seems sensitive to pinching, etc. Optic nerves normal.

Temporary improvement under KI. and syr. ferri iodidi.

April 27th, second ophthal. exam. Slight neuritis with some swelling (no "stauung") Headaches. In middle of May it was noticed that patient held her head obliquely to the left. Exam. on 20th revealed left homonymous hemianopsia. This symptom was the only one indicating a focal lesion of the brain, and it persisted. Death in June, of broncho-pneumonia.

Autopsy.—Two tumors were found in the brain; one at the apex of the left frontal lobe, the other near the apex of the right occipital lobe. Ependyma of ventricles granular; slight thickening of pia over chiasma and in both fossæ Sylvii.

The second tumor lay in the mesal aspect of the right occipital lobe, projecting a few mm. above the level of the brain, firmly adherent to the pia and only slightly to the

dura. Its length was 3 cent., height 3 cent., thickness 2.5 cent.—mostly buried in brain substance. It lay directly over the sulcus hippocampi, extending to either side of it. Basis of occip. lobe not involved. Tumors caseous.

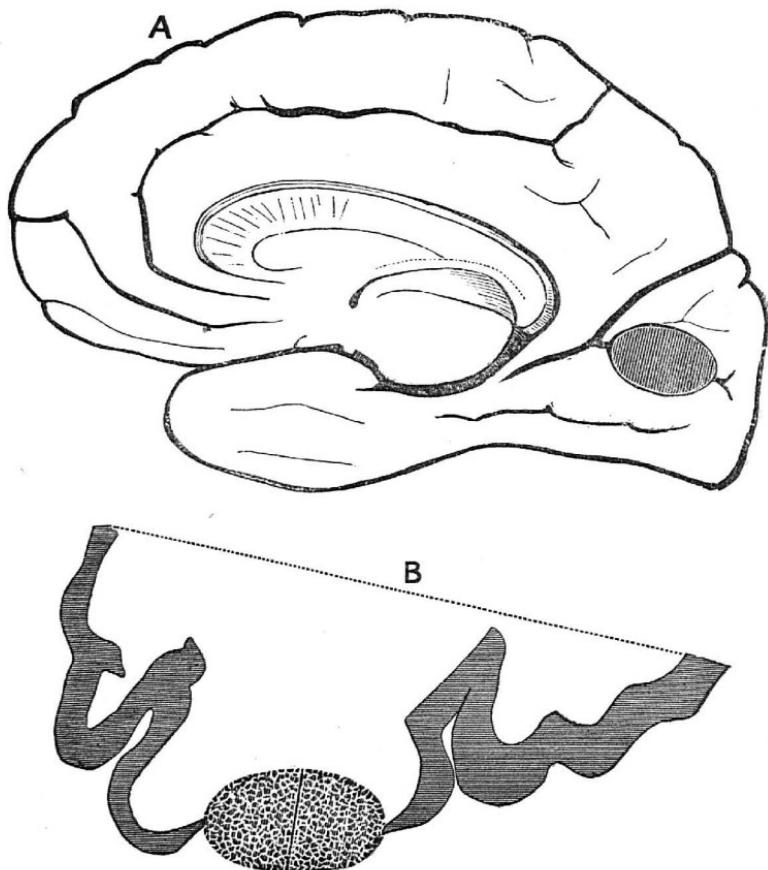


FIG. 3.—A. Tumor in mesal aspect of right occipital lobe : left lateral hemianopsia. B. Diagram of horizontal section, showing the slight penetration of the tumor. Case 29 (Huguenin).

CASE 41.—Féré. Female, æt. fifty-two. In November, 1883, sudden apoplectic attack followed by transient right hemiplegia. On admission to the Salpêtrière, no motor symptoms. Partial and slight right hemianesthesia to cold and pain. Hearing, taste, and smell normal. Typical right lateral hemianopsia, vertical line

passing through point of fixation. No ophthalmoscopic lesions; state of pupils not mentioned.

Death Dec. 24, 1884. Autopsy showed only a yellow patch destroying the greater part of the left cuneus and encroaching somewhat on the adjacent second temporal gyrus (gyr. temp. 5 of Ecker). No secondary degeneration. Corpora geniculata, lobi optici, tractus, chiasm, and optic nerves normal. Gray commissure of 3d V. absent.

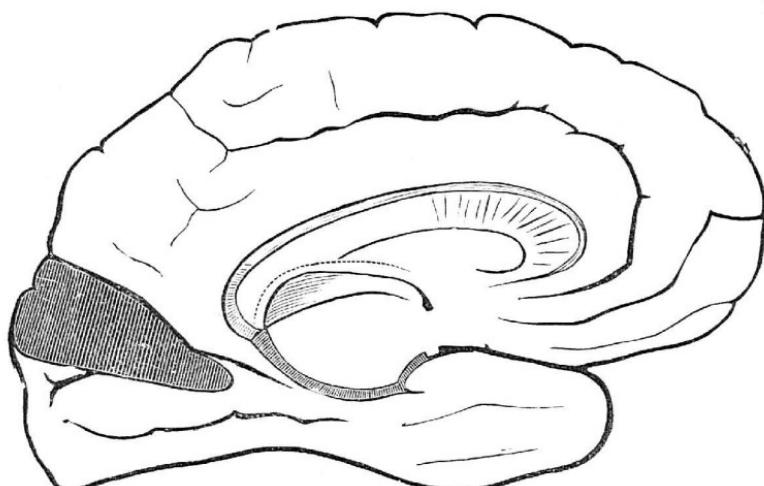


FIG. 4.—Mesal aspect of left hemisphere (Ecker). Patch of softening causing right lateral hemianopsia. Case 41 (Férec).

CASE 45.—Seguin. Mr. J. W. D., æt. 46, consulted me on January 18, 1884, for insomnia and dyspepsia. Wakefulness was most marked in early morning. Has grown paler, weaker, and thinner of late. Denies dyspncea. Examination showed general anaemia, feeble, slow pulse (63 to 66 beats per minute). Heart feeble, with a distinct, harsh, mitral regurgitant murmur. There were pulsations in the external jugular veins. Urine normal, though of high specific gravity.

Under digitalis, cannabis indica, nux vomica, and arsenic, in various combinations, and a much more nutritious diet, with a glass of rich claret at his meals, a good recovery was obtained in about six weeks. Sleep was sound; the patient had regained weight and color.

November 26, 1884, I was sent for to visit him at his home. I learned that in the spring he had travelled south as far as Havana, and returned in excellent health, to all appearances. He was then, and for the succeeding three or four months, much overworked, and especially worried about his business, which was far from

prosperous. He had given up his claret, and, most unfortunately, purchased and used quite actively a rather strong home-gymnasium. His house was situated at the top of one of our hilliest streets, and this he climbed rapidly every day.

I found him suffering apparently from regular intermittent fever; severe chills followed by high fever and sweating. He had been severely purged, and was quite weak. His heart was larger than when first seen, and the mitral murmur was much louder and more diffused. He was given quinine and nutritious food.

A few days thereafter, about December 5th, I was sent for in haste, because of an attack of a nervous nature. I found Mr. D. considerably alarmed, but rational, and free from serious symptoms. He complained of numbness in the whole left side, cheek, arm, leg, and trunk; most marked in the hand and foot. There was no distinct hemiplegia, and no anaesthesia to ordinary tests; he thought, however, that tactile sensibility, as tested by passing his fingers over objects, was somewhat duller. He was most concerned, however, about another symptom, which he stated as a "blindness of the left eye." He could not, he said, see objects on his left without turning his head and eyes. Testing by means of a small bright object in the usual way, revealed typical left lateral hemianopsia, with a vertical division line not including the points of fixation. Central vision was as good as ever, as tested by newspaper type. Dr. C. R. Agnew was asked to see the patient the next day, and the following is a copy of his report of the condition of the patient's eyes:

"My dear Doctor: I have examined Mr. D.'s eyes. He has left hemiopia, as you say. He has opaque nerve fibres in nasal half of left optic disk, extending off a little distance into the fundus, which is physiological. He has a few punctate changes in the pigment layer of retina in *both* eyes, chiefly the left. I do not think that these things have any thing to do with the eye trouble—that is *central*, as you say. I agree with you in all you say, and have nothing to suggest in the way of topical treatment.

"Yours faithfully, C. R. AGNEW."

My diagnosis at the time was embolism of a branch of the posterior central artery supplying the meso-caudal part of the right occipital lobe.

Mr. D.'s illness lasted, with most remarkable symptoms and extraordinary remissions, until May 17, 1885, when he died.

The chief features of this long sickness may be summarized as follows:

In December he had a violent attack of acute hallucinatory mania (both aural and visual hallucinations), due probably to cerebral anaemia. Under large doses of chloral, digitalis, and most persistent feeding with large quantities of milk and eggs, this subsided.

In February Mr. D. was able to go to Nassau, N. P. While there the severe chills, high fever, and sweats returned, and

proved rebellious to large doses of quinine. These chills followed no distinct type of periodicity ; they occurred twice a day, every second day or daily.

He returned to New York April 5th, and to the last, recurring febrile paroxysms usually clearly intermittent, were prominent features of the case. His general condition was better, but the heart was larger and presented an extremely loud and diffused mitral regurgitant murmur. During the month several attacks of visceral and peripheral embolism occurred, characterized by haematuria, splenic pain, and enlargement, a few discolored patches under the skin.

[In the preceding November, shortly after the hemianopsia, he one day complained of pain and swelling of the right palm, followed by a turgid condition of the whole hand for several days ; probably embolism of a part of the palmar arch.]

These embolisms were all recognized at the time as dependent upon the mitral disease, and it finally occurred to me that the intractable, irregular intermittent fever might also be of cardiac origin, each attack due to the detachment of microscopic particles of the diseased valves.

Dr. William H. Draper was called in consultation May 8th, and made the formal diagnosis of ulcerative or malignant endocarditis.

Previous to death, for a period of about a fortnight, the patient's speech was sometimes difficult to understand ; his articulation was defective, partly from extreme general debility, but also from some want of power in the buccal muscles. The hands both showed disorders of movements, choreiform tremors, and in the left hand slight ataxia in larger motions.

Often Mr. D. complained of numbness and coldness of the left hand.

At no time was there distinct hemiplegia or monoplegia, and repeated tests of sensibility showed it to be nearly if not quite normal, so that I was of the opinion that no emboli of any size had reached the brain since the attack in November. Several tests were made of the hemianopsia, one a few days before death. It persisted to the last unchanged, as judged by rough measurement, and vision remained good. The patient always insisted that his left eye was weak (a statement made by other hemianopsic patients). He was able to read and write easily until a few weeks before death, when increasing debility confined him to his bed.

The autopsy was made with the assistance of Dr. W. R. Birdsell, on the evening of the fatal issue, May 17th. The spleen and kidneys contained several infarcts of various ages, some very large, and looking like hemorrhagic foci.

The heart was much enlarged ; the mitral valves deformed and bearing enormous rough vegetations, one almost polypoid. Sections through some of these vegetations, stained by Gram's method, showed under the microscope globular nests of micrococci and separate colonies of bacteria. The aortic valves and aorta were normal.

The brain was generally anæmic. The basal vessels and middle cerebral arteries free from emboli and thrombi. The basal nerves, the optic tracts, and the chiasm were most carefully examined and found normal. On the right lateral aspect of the pons, caudad of the IVth nerve, a small branch of the basilar artery contained a firm thrombus of dark red color about 4 mm. in length; the vessel supplied the velum medullare anterius. The left hemisphere presented a small area of extreme congestion and ecchymosis over the folds of the second frontal gyrus; there was another patch at the foot of this gyrus extending toward the orbital gyri.

The right hemisphere had a similar superficial recent lesion (ecchymosis) at the vertex, extending over the dorsal extremity of the fissure of Rolando.



FIG. 5.—Mesal aspect of right hemisphere (Ecker). Patch of softening causing left lateral hemianopsia. Case 45 (Seguin).

Viewing the brain from above, the occipital extremity of the right hemisphere appears thinner than its homologue. This is found to be due to the destruction of the mesal surface of the right occipital lobe by a large focus of yellow softening, evidently an old patch. The lesion involves the basal part of the cuneus, the fourth and fifth temporal gyri (Ecker), and a part of the gyrus hippocampi. The destruction does not quite attain the tip of the occipital lobe.

The remaining gyri of both hemispheres were normal.

I would add that the above records of the appearance of the brain were made at the time of examination by our President, Dr. Birdsall, and me.

Most unfortunately, the brain was not cut at once. The left hemisphere was separated, leaving the "stamm" attached to the right hemisphere, and these were placed in bichromate of potassium, with the intention of making a series of sections after complete hardening.

Through mishaps the process of hardening was not very successful, and the right hemisphere particularly suffered from too prolonged pressure upon its temporal lobe, which disintegrated. The result is that I can only show you to-night the occipital half of the right hemisphere with the patch, which I consider the essential and truly causal lesion of the hemianopsia. The destruction of tissue extends only a few mm. into the subjacent white substance. The state of the internal capsule, thalami, etc., remains unknown, owing to the misfortune in preserving the specimens. From the history of the case, however, judging from the absence of hemiplegia and marked anaesthesia, it may be safely assumed, in the light of our present pathological knowledge, that there were no lesions, or at least no tangible lesions, in the central parts of the brain.

That the destruction of the right cuneus and fifth temporal gyrus was the cause of the left lateral hemianopsia during life, I have not a shadow of a doubt.

The softening was produced by embolism of the third branch of the posterior cerebral artery, the occipital artery of Duret.

The objections which may be presented against the value of my case, in consequence of its imperfect anatomical investigation, are greatly reduced in force by the consideration that the case is one in harmony with many others. Were it a contradictory or anomalous case, it would certainly possess much less value.

Now, gentlemen, what conclusion may reasonably be drawn from all these cases?

1. That lesions in the mesal aspect of the temporal lobes, or even in other basal districts of the hemispheres, may give rise to hemianopsia indirectly by pressing upon the primary optic centres or upon the tractus optici and chiasm.

2. That lesions of the corpus geniculatum laterale, pulvinar, and latero-caudal parts of the thalamus may cause hemianopsia; usually in association with hemianæsthesia and hemiplegia, or hemianæsthesia alone.

3. That a lesion of the white substance of the occipital lobe, in the caudal radiations of the internal capsule, may cause hemianopsia alone, or with hemianæsthesia.

4. That lesions of the supra-marginal gyrus, angular gyrus, and inferior parietal lobule with the subjacent white substance may cause hemianopsia—with or without other symptoms (hemiplegia, loss of muscular sense, word-deafness, etc.).

5. That a lesion of greater extent, involving the speech centre, the motor convolutions, and the parts enumerated above (4), due usually to embolism or thrombosis of the entire Sylvian artery, will, when existing on the left side, produce aphasia, alexia, hemianopsia, and hemiplegia.

6. That lesions of the occipital lobe, cortex, and subjacent white matter produce blindness when bilateral, and hemianopsia when unilateral. This conclusion is in accord with Exner's (1881).

7. That a lesion of the cuneus and adjacent 5th temporal gyrus (Ecker) on one side produces lateral hemianopsia of the opposite side.

In support of this last conclusion I would again invite your attention to the cases 28, 29, 41, and 45.

I have endeavored to fuse the diagrams of the sixteen cases with occipital lesions (exclusive of the traumatic ones) on one chart, by the successive application of layers of India ink. The larger lesions were first indicated on the outline diagram, and the most limited lesions washed last. I was somewhat hindered by the "running" of the black in the lines indicating gyri, yet I think that the maximum color due to the superposition of the greatest number of layers is over the cuneus, and next to the occipital apex, as a whole. This is a simplification of one of Exner's methods, and I think may, with some improvements, be made serviceable for clinical teaching. [The diagram was shown at the time of reading the paper.]

Let us now turn to the physiological and theoretical aspect of our subject. My time is limited, and I can only treat this highly important matter in a most summary way.

What do the most modern physiological researches teach us upon this question of the location of the cortical visual centre and its white connecting fasciculi?

The views of Munk and Ferrier are the authoritative ones.

The former physiologist has persistently taught that the visual areas, or centres for psychic vision, are in the occipital lobes, and that each visual area has connections with both retinæ. He invariably produced hemianopsia in dogs by destruction of one occipital lobe. These experimental results have been verified by Ganser, von Gudden's assistant, upon kittens.

Ferrier's theory, supported, as he believed, by experiments upon monkeys, has received an apparent verification at the hands of Prof. John C. Dalton.¹ Ferrier thought that the visual centre was in the angular gyrus. The following are his most recent conclusions, as presented to the Royal Society, and published in its Proceedings, xxxv., p. 229, and abstracted in *Brain*, April, 1884.

1. Lesions of the occipito-angular region (occipital lobes and angular gyrus) cause affections of vision without affection of the other sensory faculties or motor powers.
2. The only lesion which causes complete and permanent loss of vision in both eyes is total destruction of the occipital lobes and angular gyri on both sides.
3. Complete extirpation of both angular gyri causes for a time total blindness, succeeded by lasting visual defect in both eyes.
4. Unilateral destruction of the cortex of the angular gyrus causes temporary abolition or impairment of vision in the opposite eye—not of a hemiopic character.
5. Deep incisions may be made in both occipital lobes at the same time, or the greater portion of one or both occipital lobes at the same time may be removed without any appreciable impairment of vision.
6. Destruction of the occipital lobe and angular gyrus on one side causes temporary amblyopia of the opposite eye and homonymous hemianopsia of both eyes toward the side opposite the lesion.
7. As in none of the cases recorded, either of partial unilateral or bilateral destruction of the occipito-angular region, were the amblyopic or hemianopic symptoms permanent, it is concluded that vision is possible with both eyes if only portions of the visual centres remain intact on both sides.

¹ John C. Dalton, in *New York Medical Record*, Oct. 26, 1881.

It will be seen that the results of our pathological analysis are seemingly favorable to both the theories of Munk and of Ferrier. But, on the one hand, the most conclusive cases, *i. e.*, those with the most limited cortical lesions, are wholly opposed to Ferrier's views and in favor of Munk's; and, on the other hand, a peculiarity in the anatomy of the occipital extremity of the brain goes to explain Ferrier's results without assuming the existence of a cortical visual centre in the angular gyrus. It is this: that the optic fasciculus of Gratiolet and Wernicke, on its way from the caudo-lateral aspects of the thalamus, in the internal capsule, passing out caudad, lies latero-dorsad of the posterior horn of the lateral ventricle, and close under the inferior parietal lobule and the angular gyrus, on its way to the occipital lobe (cuneus chiefly). A lesion of the angular gyrus, the supra-marginal gyrus, and even of the inferior parietal lobule, is almost certain to involve this optic fasciculus, and thus cut the communication between the visual centre and the eyes.

I pass around a specimen in which, on a horizontal longitudinal section of the human brain hardened in bichromate of potassium, the optic fasciculus is plainly visible as a homogeneous whitish band. It is evident that lesions in the angular gyrus and supra-marginal gyrus could easily penetrate deeply enough to injure this fasciculus.

It seems to me that with this anatomical knowledge the discrepancies between Ferrier and Munk's results disappear in part, and that some of the cases of my sixth category (cases 26, 32) are reconciled with the others.

Next, as to the various purely hypothetical or clinical theories of the course of the optic paths. Of these the best known is that of Prof. Charcot. His well-known diagram of the course of the optic fibres from the retina to the visual centres represents a second decussation of the fasciculi laterales through the corpora quadrigemina (lobi optici) on their way to the internal capsule, so that finally each internal capsule contains all the fibres for the opposite eye. This diagram was made to explain and support Charcot's theory of the production of amblyopia of one eye by lesion of the occipital lobe and the internal capsule of the opposite side. He

thought that he had observed that amblyopia of one eye, and not hemianopsia, was the companion of hemianæsthesia produced by lesion of the internal capsule.

I regret to say that my illustrious master's theory has not been supported by either clinical observations or by post-mortem results. I know of but one case with a post-mortem examination which is in favor of Charcot's view,¹ while the sixteen cases I have read to you speak emphatically against it. Indeed, there is reason to believe that Prof. Charcot has never attached much value to his diagram, and I understand that he has already abandoned it, yielding, as he is ever ready to do, any theoretical views of his own to opposing pathological facts.

Grasset has recently (1883) offered a modification of Charcot's diagram, which is extravagant. He would have still a third decussation (counting the chiasmic as the first) somewhere in the callosal fibres, so that after the fibres for one whole retina, according to Charcot's schema, have passed a certain distance in the internal capsule, the fibres of the fasciculus lateralis again cross the median line, so that the visual centre receives fibres from both retinæ. This far-fetched attempt to reconcile Charcot's opinion as to the effect of lesion of one internal capsule in its caudal division, with the well-established results of lesions of the occipital lobes, is hardly deserving of serious criticism; but it may be as well to state that more recent (1884)² experiments by W. Bechterew show that in dogs at least section of the posterior part of the internal capsule produces lateral hemianopsia—a result in full harmony with some of our human cases.

From his latest pathological observations von Monakow³ draws the following conclusions as to the course of the central optic fasciculi in man:

"The collective optical bundle forms a solid tractus in the sagittal white substance of the occipital part of the brain, which passes alongside of the corpus callosum fibres or tapetum, and ends in the cortex of the occipital gyri,

¹ Petrina, in *Prager Zeitsch. f. Heilk.*, II., p. 595, case viii. *Vide* Table I.

² W. Bechterew: Ueber die nach Durchschneidung der Sehnervenfasern im innere der Grosshirnhemisphären, etc. *Neurol. Centralbl.*, 1884, No. 1.

³ *Westphal's Archiv f. Psychiatrie*, xvi., 352.

more especially in that of the cuneus, lobus lingualis, and gyrus descendens."

The diagram of optic paths which I offer you is, I believe, in agreement with Munk's view of the physiology of the visual centre, with what we know of the anatomy of the optic tracts by dissection and by secondary degeneration (Monakow), and lastly, best of all, with the results of now numerous post-mortem examinations.

From the above data, pathological, anatomical, and experimental, are we now in a position to induce diagnostic laws with reference to the symptom hemianopsia? I think we are, and I would propose the following as a preliminary set of rules.

1. Lateral hemianopsia always indicates an intra-cranial lesion on the opposite side from the dark fields.
2. Lateral hemianopsia with pupillary immobility, optic neuritis or atrophy, especially if joined with symptoms of basal disease, is due to lesion of one optic tract, or of the primary optic centres on one side.

This diagnosis may be further strengthened and rendered quite certain by seeking for and finding one-sided pupillary reaction, as recently suggested by Wernicke.¹ He ingeniously predicts that only one lateral half of each iris will be found to contract by the reflex effect of light when one optic tract has been interrupted. He designates this as "hemiopic pupillary reaction."

3. Lateral hemianopsia, or sector-like defects of the same geometric order, with hemianæsthesia and choreiform or ataxic movements of one half of the body without marked hemiplegia, is probably due to lesion of the caudo-lateral part of the thalamus, or of the caudal division of the internal capsule.

4. Lateral hemianopsia, with complete hemiplegia (spastic after a few weeks) and hemianæsthesia, is probably caused by an extensive lesion of the internal capsule in its knee and caudal part.

5. Lateral hemianopsia, with typical hemiplegia (spastic

¹ Wernicke: Ueber hemiopische Pupillenreaction. *Fortschritte der Medicin*, 1883, i., 49-53.

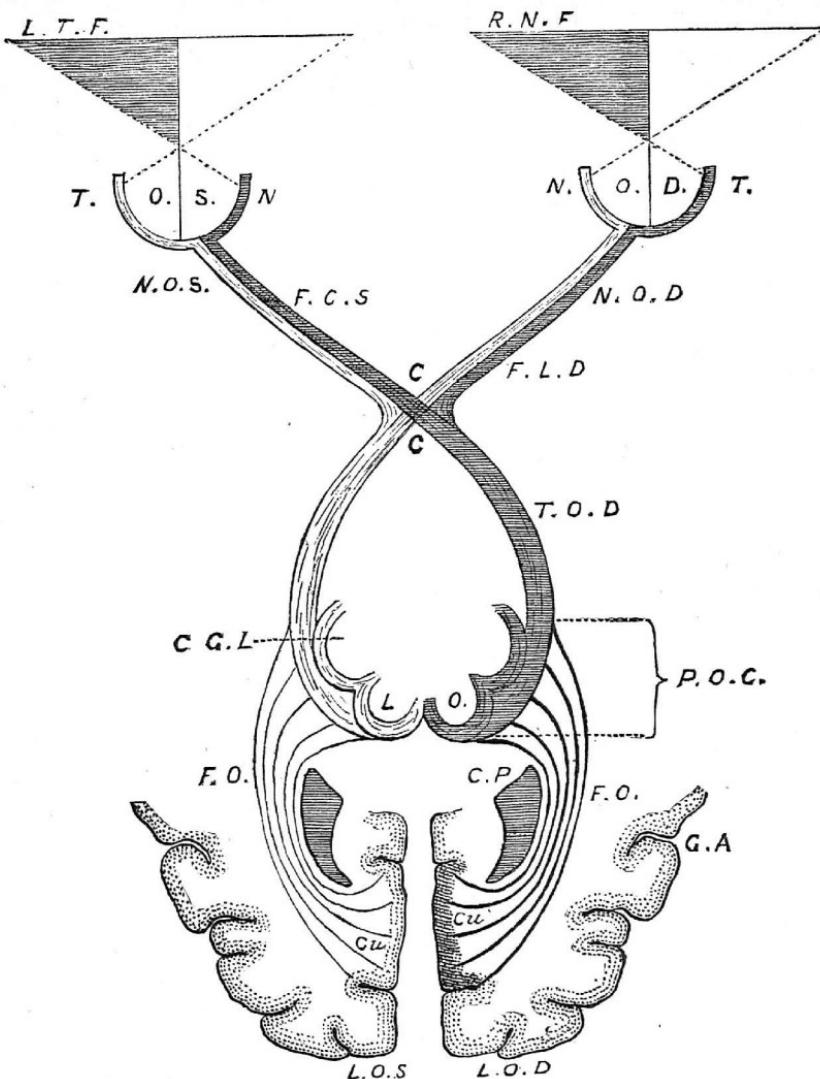


FIG. 6.—Diagram of Visual Paths ; designed to illustrate specially Left Lateral Hemianopsia from any lesion. L. T. F., left temporal half-field. R. N. F., right nasal half-field. O. S., oculus sin. O. D., oculus dexter. N. T., nasal and temporal halves of retinæ. N. O. S., nervus opticus sin. N. O. D., nervus opticus dext. F. C. S., fasciculus cruciatus sin. F. L. D., fasciculus lateralis dext. C., chiasma, or decussation of fasciculi cruciati. T. O. D., tractus opticus dext. C. G. L., corpus geniculatum laterale. L. O., lobi optici (corpus quad.). P. O. C., primary optic centres, including lobus opticus corp. genic. lat., and pulvinar of one side. F. O., fasciculus opticus (Gratiolet) in the internal capsule. C. P., cornu posterior. G. A., region of gyrus angularis. L. O. S., lobus occip. sin. L. O. D., lobus occip. dext. Cu., cuneus and subjacent gyri constituting the cortical visual centre in man. *The heavy or shaded lines represent parts connected with the right halves of both retinæ. The reader may place the lesion as he pleases.*

after a few weeks), aphasia if the right side be paralyzed, and with little or no anæsthesia, is quite certainly due to an extensive superficial lesion in the area supplied by the middle cerebral artery; we would expect to find (as in case 26, by Westphal) softening of the motor zone and of the gyri lying at the extremity of the fissure of Sylvius, viz.: the inferior parietal lobule, the supra-marginal gyrus, and the gyrus angularis. Embolism or thrombosis of the Sylvian artery would be the most likely pathological cause of the softening.

6. Lateral hemianopsia with moderate loss of power in one half of the body, especially if associated with impairment of muscular sense, would probably be due to a lesion of the inferior parietal lobule and gyrus angularis, with their subjacent white substance, penetrating deeply enough to sever or compress the optic fasciculus on its way caudad to the visual centre.

7. Lateral hemianopsia without motor, or common sensory symptoms, this symptom alone, is due, I believe, from the convincing evidence afforded by Cases 28, 29, 41, and 45, to lesion of the cuneus only, or of it and the gray matter immediately surrounding it on the mesal surface of the occipital lobe, in the hemisphere opposite to the dark half-fields. Most surgical cases come at once, or after convalescence, within this rule or in No 6 (Case 3.)

In all cases coming under rules 3 to 7 inclusive, the pupils react normally; and rarely does the ophthalmoscope show any lesion of the optic nerve, except, of course, in some tumor cases, when neuro-retinitis may be expected.

A LIST OF CASES OF HEMIANOPSIA OF CENTRAL ORIGIN, WITH AUTOPSY, AND OF TRAUMATIC HEMIANOPSIA, TO OCTOBER 30, 1885, ARRANGED IN CHRONOLOGICAL ORDER.

1, 1863. CHAILLOU. Société anatomique, Feb., 1863. *Bull. de la Soc. Anat.*, 1863, p. 71.

2, 1866. LEVICK. Case of abscess of the brain. Trans. of the College of Physicians of Phila., March 7, 1866. *Am. Journal Med. Science*, 1866, ii., p. 413.

3, 1871. W. W. KEEN and WM. THOMSON. Gunshot wound of the brain, followed by fungus cerebri, and recovery with hemiopia. *Trans. Am. Ophthal. Soc.*, 1871, p. 122. Also in "Medical and Surgical History of the War of the Rebellion," i., p. 206.

4, 1873. HUGHES, J. S. Case of compound depressed fracture of the skull. *Irish Hospital Gazette*, July 1, 1873 (abst. in *Nagel's Jahresbericht über Ophthalm.*, 1874, p. 440).

5, 1875. HIRSCHBERG, J. Zur Semidecussation der Sehnervenfasern in Chiasma des Menschen. *Virchow's Archiv*, lxv., p. 116.

6, 1875. JACKSON, J. H., and GOWERS, W. R. I. A case of hemiopia, with hemianesthesia and hemiplegia, by Dr. Jackson. *Lancet*, 1874, ii., 306. II. Autopsy on a case of hemiopia, etc., by Dr. Gowers. *Lancet*, 1875, i., 722.

7, 1876. FÖRSTER and WERNICKE. Förster in "Graefe-Saemisch Handbuch der gesammten Augenheilkunde," Bd. vii, p. 118. Also in Wernicke's "Die aphatische Symptomcomplex," p. 47. Berlin.

8, 1876. HUGUENIN. Article "Chronic Abscess of the Brain," in Am. ed. of *Ziemssen's Cyclop.*, xii., 796.

9, 1877. POOLEY, T. R. Right-sided binocular hemiopia, depending upon a gummy tumor in the left posterior lobe of the brain. *Knapp's Archives of Ophthalm.*, v., p. 148; ix., p. 83.

10, 1877. JASTROWITZ, M. Tumor im linken Hinterlappen, Aphasie, rechtsseitige Hemianopsie. *Hirschberg's Centralbl. f. pract. Augenheilkunde*, 1877, p. 254.

11, 1877. JASTROWITZ. Case referred to briefly in his paper in *Hirschberg's Centralbl. f. pract. Augenheilk.*, i., p. 256.

12, 1877. CHARCOT et PITRES. Contributions à l'étude des localizations dans L'écorce des hémisphères cérébraux. *Revue mensuelle de méd. et de chir.*, i., p. 372, obs. xxxiv. (1877).

13, 1878. BAUMGARTEN, PAUL. Hemiopie nach Erkrankung der occipitalen Hirnrinde. *Centralbl. f. Med. Wissenschaft*, 1878, No. 21.

14, 1878. HOSCH, FR. Zur Lehre von der Sehnervenkreuzung. *Zehender's Monatsblätter f. Augenheilk.*, 1878, p. 281.

15, 1879. DMITROWSKY and LEBEDEN. Hemianopsia dextra. Monoplegia facialis dextra. Hæmorrhagia und Erweichung in der linken Hirnhalbkugel. *Medicin. Bote*, 1879, No. 46. (Abst. in *Hirschberg's Centralbl.*, 1880, p. 84.)

16, 1879. PFLÜGER. Augenklinik in Bern. *Bericht über das Jahr* 1878, p. 57.

17, 1879. NOTHNAGEL. Topische Diagnostik der Gehirnkrankheiten. Berlin, 1879, p. 389.

18, 1879. CURSCHMANN. In Berliner Gesellschaft f. Psych. und Nerven-krankheiten, Juni 9, 1879. *Westphal's Archiv*, xi., 822.

19, 1880. DRESCHEFELD. Pathologische-anatomische Beiträge zur Lehre von der Semidecussation der Sehnervenfasern. Case ii. *Hirschberg's Centralbl. f. pract. Augenheilkunde*, 1880, p. 35.

20, 1880. SCHMIDT-RIMPLER. Sitzb. der ärztl. Verein zu Marburg, Juni 9, 1880 (abst. in *Hirschberg's Centralbl.*, 1881, p. 358).

21, 1881. LINNELL, E. H. A case of left-sided binocular hemianopsia, with report of autopsy and microscopical examination. *Knapp's Archives of Ophthalm.*, x. (1881), 4, p. 446.

22, 1881. WESTPHAL, C. Zur Frage von der Localization der unilaterale Convulsionen und Hemianopsie. *Charité-Annalen*, vi., 1881, p. 342.

23, 1881. HEUSE. Hemianopsie bei Schädelverletzung. Case ii. *Hirschberg's Centralbl. f. pract. Heilkunde*, 1881, p. 205.

- 24, 1881. SENATOR, H., in WERNICKE'S Lehrbuch der Gehirnkrankheiten, ii., p. 70, 1881.
- 25, 1881. PETRINA. Ueber Sensibilitätsstörungen bei Hirnrindenlesionen. Case viii. *Zeitschr. f. Heilkunde*, Prag, 1881, ii., 375.
- 26, 1882. WESTPHAL, C. Zur Localization der Hemianopsie und des Muskelgefühls beim Menschen. *Charité-Annalen*, Bd. vii., 1882, p. 466.
- 27, 1882. MARCHAND. Beitrag zu Kenntniß der homonymen bilateralen Hemianopsie, und der Faserkreuzung in Chiasma opticum. *Archiv f. Ophthal.*, xxviii., 2, 63. Case iii.
- 28, 1882. HAAB, O. Ueber Cortex-Hemiopie. *Klinische Monatsblätter f. Augenheilkunde*, xx., 141, 1882.
- 29, 1882. HUGUENIN. Case in Haab's article.
- 30, 1882. DRESCHFELD, J. Pathological contributions to the course of the optic nerve fibres in the brain. *Brain*, iv., p. 543, Jan., 1882. Case i.
- 31, 1882. *Idem*. In *idem*. Case ii.
- 32, 1882. STENGER, C. Die cerebralen Sehstörungen der Paralytiker. *Arch. f. Psych.*, xiii., p. 242. Case vii.
- 33, 1882. *Idem*. In *idem*, p. 246. Case viii.
- 34, 1882. WERNICKE and HAHN. Idiopathischer Abscess des Occipitallappens, durch Trepanation entleert. *Virchow's Archiv*, lxxxvii., 335.
- 35, 1883. JANY, L. Ein Fall von rechtsseitiges Hemianopsie und Neuro-retinitis in Folge eines Gliosarcoms im linken occipitallappen. *Knapp's Archiv f. Ophthal.*, xii., p. 327.
- 36, 1883. ROSENBACH, P. Zur Casuistik der Hemianopsie. *St. Petersburg Med. Wochenschr.*, 1883, No. 12. *Neurolog. Centralbl.*, 1883, p. 442.
- 37, 1883. NIEDEN, A. Ein Fall von einseitiger temporaler Hemianopsie des rechten Auges nach Trepanation des linken Hinterhauptbeins. *Archiv f. Ophthal.*, xxix., iii., p. 143.
- 38, 1883. RICHTER. Fall von dreimaliger Blutung in ein Gehirn. Gesell. f. Psych. u. Nervenkrankheiten, 11 Juni, 1883. *Neurolog. Centralbl.*, 1883, p. 307.
- 39, 1883. SCHMALTZ. Case given by A. Vetter; Ueber die sensorielle Function des Grosshirns, etc. *Deutsch. Archiv f. kl. Medicin*, xxxii., p. 469, 1883.
- 40, 1884. WIETHE, TH. A case of superior homonymous hemianopia. *Knapp's Archives of Ophthal.*, xiii., 3, 301.
- 41, 1885. FÉRÉ, CH. Trois autopsies pour servir à la localisation cérébrale des troubles de la vision. Case iii (from Charcot's service). *Archives de Neurologie*, Mars, 1883, p. 229.
- 42, 1885. RICHTER, A. Zur Frage der optischen Leitungsbahnen des menschlichen Gehirns. *Archiv f. Psych. und Nervenkrankheiten*, xvi., p. 641.
- 43, 1885. *Idem*. Ueber die optischen Leitungsbahnen des menschlichen Gehirns. Fall ii. *Allg. Zeitschr. f. Psych.*, xli., 1885, p 637.
- 44, 1885. *Idem*. In *idem*. Fall iii., p. 638.
- 45, 1885. SEGUIN, E. C. A contribution to the pathology of hemianopsia of central origin. Case with specimen. Read before the New York Neurological Society, October 6, 1885.